Motor Unit Structural Change Post Stroke Examined via Surface Electromyography: A Preliminary Report*

Brian Jeon, Nina L. Suresh, Aneesha K. Suresh, William Z. Rymer, and Xiaogang Hu

Abstract—Muscular weakness is one of the major impairments limiting motor function following a hemispheric stroke. The objective of this preliminary study was to examine possible motor unit (MU) structural changes in paretic muscle post-stroke as a measure by which to assess neural and/or biomechanical mechanisms of paresis. A surface electromyogram (sEMG) recording and decomposition system was used to record sEMG signals and extract single MU activities from the first dorsal interosseous muscle (FDI) of three hemiparetic stroke survivors. To characterize potential MU structural changes, an estimate of the motor unit action potential (MUAP) amplitude and duration was derived using the spike triggered averaging of the sEMG signal. Our preliminary results reveal MUAPs with systematically smaller amplitude and longer duration in the paretic muscle compared with the contralateral muscle of three tested stroke subjects with varying degrees of motor impairment. The changes in MU properties such as reduced MU size and a reduction in the muscle fiber conduction velocity could contribute at least in part, to muscle weakness post-stroke. The sEMG recording and decomposition system combined with our spike triggered averaging technique has the potential to provide an assessment tool for muscular weakness post-stroke.

I. INTRODUCTION

Cerebral stroke is a leading cause of disability in the United States [1]. After a stroke injury, muscular weakness is one of the major impairments limiting motor function in stroke survivors [2–4]. Possible mechanisms of weakness include reduced excitatory descending drive, muscle atrophy, and disturbance in the control of the MU pool [5–9]. However, the choice as to which mechanisms should be targeted during therapeutic intervention is still unclear, partly due to the insufficient understanding of relative impact of these particular mechanisms [10–12].

Only a few studies have investigated disturbances of MU structural properties, using parameters such as MU size and muscle fiber conduction velocity in paretic muscles of stroke survivors [5, 13, 14] as a means by which to understand the underlying mechanism of weakness. A reduction in MU size could lead to a reduced MU twitch force. Therefore, to achieve any desired overall muscle force, more MUs, possibly more fatigable, need to be activated, which ultimately can lead to muscle weakness. A reduction in fiber conduction velocity could indicate changes in overall distribution of motor unit type as well as changes in the recruitment pattern of motor units. Indeed, earlier studies have shown that the large and fast MUs are more selectively affected compared with small and slow MUs post-stroke [15, 16].

Typically, MU structural changes are assessed using invasive intramuscular EMG recordings or biopsy samplings [13, 16] and the data are collected piecemeal. Thus, a non-invasive and systematic examination of MU structural change post-stroke is necessary to extend our understanding of the neural mechanisms of muscle weakness. A novel sEMG electrode array recording and decomposition method has recently been developed by De Luca and colleagues [17, 18] and it yields a large number of MUs simultaneously over a relatively large force range. Using the results from this sEMG decomposition technique, we have developed an analytical method to examine MUAP properties [19, 20]. Using these novel techniques, the objective of this preliminary study was to examine the change in MU structure to understand the contributions to muscle weakness during voluntary muscle activation post-stroke.

II. METHODS

A. Subjects

Three chronic hemiparetic stroke subjects with varying degrees of weakness (see Table 1) of the extremities contralateral to the cerebral lesion were tested. All participants gave informed consent via protocols approved by the Institutional Review Board at Northwestern University.

<table>
<thead>
<tr>
<th>ID</th>
<th>Sex</th>
<th>Age</th>
<th>Side</th>
<th>Time</th>
<th>Chedoke</th>
<th>FM</th>
<th>MVC</th>
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<td>R</td>
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<td>F</td>
<td>59</td>
<td>R</td>
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<td>M</td>
<td>61</td>
<td>R</td>
<td>4</td>
<td>6</td>
<td>63</td>
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B. Experimental Setup

The subjects were seated in a Biodex chair with their forearm fixed with a brace and a ring mount to restrain unnecessary movement and minimize the effect of unrecorded muscle activity (Fig. 1A). The elbow of the subject was also comfortably resting on a support during the experiment. The proximal phalanx of the index finger was fixed to a six degrees-of-freedom load cell (ATI, Inc.) while keeping the
entire index finger aligned with the long axis of the forearm. The load cell recorded the forces generated by the subject in the x-direction (abduction/adduction). The recorded force was low-pass filtered at cutoff frequency of 200 Hz, and digitally sampled at 1 kHz.

The sEMG was recorded from the first dorsal interosseous (FDI) of both affected and contralateral side of each subject. A Delsys surface sensor array (Fig. 1B and 1C), which is composed of five probes (one probe at the center and four probes around the center probe in a 5 mm x 5mm square pattern), was used to obtain the sEMG recording. The recordings from the 5 electrodes were then differentiated to obtain 4 different channels. The four channels are band-pass filtered at 20 Hz to 2000 Hz with a sampling rate of 20 kHz.

Fig. 1: (A) Experimental setup, (B) Sensor array placement, (C) Sensor array probes, (D) sEMG recording from 4 channels and an enlarged segment of the sEMG recording during a steady state contraction. (E) Force trajectory of each trial.

**C. Procedures**

Subjects were tested two different times, less than a week apart from each other, to test both affected and contralateral sides of the hands using the same procedure. Before the trials began, the subject’s maximum voluntary contractions (MVCs) of both sides were measured. The MVC of the affected side was used to determine the force levels for both the contralateral and the affected side for fair comparison of the two sides.

The trials consisted of isometric force contraction in which the subjects followed a trapezoid force trajectory as seen in Fig. 1E. The steady state force (top of the trapezoid) of each trial was determined as a percentage of MVC (i.e., 20%, 30%, 40%, 50%, and 60%), and the rise of the ramp was fixed at 10% MVC per second for all force level. Prior to the main trials, they were given five practice repetitions of 30% MVC trial so that the subjects could learn to follow the trajectory as closely as possible during the main trials. Then, the subjects started the main trials involving the five different force levels as mentioned above. Each force level was repeated four times with one-minute rest between each force level to minimize the effect of fatigue on the muscle. More resting time was provided, if requested by the subject. The order in which the different force level was tested was also randomized per subject. The same procedure was used to test both the affected and the contralateral side of the arm.

**D. Data Analysis**

For each of the subject, three trials from each of the five different force levels (15 total trials) were selected based on the quality of the sEMG and force signals, determined by the subsequent characteristics: (1) There was no sudden change in force (i.e., larger than 20% MVC/s) during the up-ramp segment of the force trajectory; (2) the force variability during the steady state force was low (less than ± 2 standard deviations of background force level); and (3) The sEMG signals had a peak-peak (P-P) baseline noise < 20 µV and signal to noise ratio > 5. Then, MUs were identified and extracted using the decomposition algorithm [17, 18] (version 1.0.0.28). The output of the decomposition system includes the event times of each MUAP and its four waveform templates (20 ms) from four different channels. Exemplar templates from the decomposition are shown in Fig. 2. The template from the affected side has lower P-P amplitude and longer P-P duration.

Fig. 2: Sample MUAP templates from the decomposition of the contralateral and affected sides of one stroke subject.

Once the selected trials were decomposed, spike triggered averaging (STA) was performed on the sEMG. STA was performed by using the firing time output from the decomposition system as the aligning point, then superimposing and averaging multiple firings of the same MU from the sEMG. This method reduces the disturbances caused by other MUs as well as the inherent noise from the recording system. STA was performed on each of the 4 channels and 4 STA MUAP estimates were computed for each MU. For each channel, a 20 ms segment centered at the firing time was used as the time interval to calculate the MUAP P-P amplitude and P-P duration. Then, the coefficient of variation (CV) and correlation coefficient were calculated to test the reliability of the identified MUs [19, 20].

First, a series of P-P amplitudes of STA MUAP was calculated using a window length of 4 s (50 – 100 spikes) of sEMG, and the window was shifted 0.5 s every step. The CV of P-P amplitude over different window segments was calculated as an estimate of the MUAP waveform variation.

Second, the correlation between STA MUAP and the decomposition MUAP was measured for additional validation. STA MUAP (20 ms) estimated over the entire
length of the trial was compared with the decomposition MUAP to calculate the correlation coefficient. In computing the correlation coefficient, the STA MUAP was shifted a maximum of 10 ms forward and backward in relation to the decomposition MUAP to find the maximum correlation between the two templates. Then, the CV and correlation coefficient were averaged between the channels to avoid bias to a certain channel. MUs with an average CV < 0.2 and an average correlation coefficient > 0.7 were used for the analysis.

In addition to the P-P amplitude, P-P duration was also computed for each all four channels of each STA MU template. After which the (probability) distributions of the P-P duration and P-P amplitude values were compared between the affected and the contralateral sides for each subject.

### III. RESULTS

Using the method delineated in the data analysis of the methods section, MUAP P-P amplitudes and P-P durations from 15 selected trials for each of the three subjects were examined. The amplitude and duration data from the 4 channels were pooled together for the data analysis. The number of MUs and the templates used in the analysis are summarized in table 2. Templates that had P-P durations > 5ms were deemed as outliers and were not included in the analysis.

<table>
<thead>
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<th>Table II.</th>
<th>MU TEMPLATE INFORMATION</th>
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<tr>
<td>Side</td>
<td>Affected</td>
</tr>
<tr>
<td>Subjects</td>
<td>MU</td>
</tr>
<tr>
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</tr>
<tr>
<td>2</td>
<td>140</td>
</tr>
<tr>
<td>3</td>
<td>298</td>
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Note: MU: number of MUs found. Temp: number of templates used for analysis. Out: number of outliers with P-P durations > 5 ms.

First, the distribution of P-P amplitude was examined for all subjects with a histogram with a marked median using the vertical line (Fig. 3A). All subjects showed smaller P-P amplitude on the affected side compared with the contralateral side. The median P-P amplitude was 0.325 mV on the contralateral side and 0.092 mV on the affected side for subject 1, 0.044 mV on the contralateral side and 0.015 mV for the affected side for subject 2, and 0.468 mV on the contralateral side and 0.276 mV on the affected side for subject 3. These between-side differences in P-P amplitudes were statistically significant (p < 0.001) using the Mann-Whitney U test. The differences in the magnitudes of P-P amplitude between subjects is dependent on many factors including the placement of the sensor array relative to the muscle and the interference caused by the thickness of the passive tissue, and therefore, inter-subject comparison was not made.

Second, the distribution of P-P duration was examined as a histogram with a marked median for all subjects (Fig. 3B). All subjects revealed longer durations for the affected side indicated by the rightward shift in the median duration, indicating possible slower conduction velocity of the muscle fiber of the affected side. For subject 1, the median duration shifted from 2.05 ms on the contralateral side to 2.45 ms on the affected side. Similarly, the median duration shifted from 2.15 ms in the contralateral side to 2.70 ms in the affected side for subject 2, and 2.00 ms on the contralateral side to 2.35 ms on the affected side for subject 3. The differences in P-P durations of the two sides of all subjects were statistically significant (p < 0.001) based on the Mann-Whitney U test.

![Fig. 3: Distribution of P-P Amplitude (A) and P-P Duration (B) of three subjects. Shown on top is subject 1, middle is subject 2, and bottom is subject 3. Vertical lines represent the median duration for each respective side.](image)

### IV. DISCUSSION

This preliminary study examines the possible contribution of the changes in MU structure to muscle weakness post-stroke, through the analysis of single MU data from both sides of two stroke subjects. A novel sEMG recording and decomposition system was used to record sEMG signals and decompose single MU activities. The system is non-invasive and yields a large number of MUs simultaneously over a large force range [17, 18]. A spike triggered averaging of sEMG was used as a second level of analysis to extract reliable estimate of MUAPs and examine MUAP properties.

The preliminary results reveal a reduction of the MUAP amplitude and an increment of the MUAP duration in the paretic side of all the three subjects, suggesting possible reductions in MU size and possible reductions in overall fiber conduction velocity post-stroke. If so, there are several possible factors that can contribute to the observed MU structural changes. The reduction of MU size can be induced either by the loss of muscle fibers or by a reduction of fiber...
diameters possibly due to disuse. The reduced fiber conduction velocity can also arise from fiber diameter reduction. The loss of muscle fibers, especially the fast twitch type II fibers as reported from biopsy studies [16, 21], can also contribute to a reduction of overall fiber conduction velocity. Motoneuron reinnervation can lead to potential fiber type changes (i.e., from fast to slow) [22], which can also induce slow conduction velocity in paretic muscles.

It is also possible that there is a thick layer of passive tissue in the paretic side, and this would increase the distance between recording electrodes and muscle fibers, which can lead to smaller and longer MUAPs. Further imaging study is necessary to examine the passive tissue properties over the muscle of the two sides in order to confirm this possible bias.

It is possible that the observed change in MUAP amplitude and duration can be an artifact of the recording technique used here. Namely, given that the sensor was placed over the skin, the small MUAP amplitudes can be a recording bias of large MUs that are deep and far away from the sensor. Likewise, the long duration can be the low-pass filtering effect of thick tissue that the potential travels through. However, an earlier study has shown that the MUs of different sizes are uniformly distributed within the FDI muscle [23]; therefore, the bias of large and deep MUs towards small ones will not hold.

Overall, the sEMG recording and decomposition system combined with the analytical STA technique will potentially provide an efficient way to identify a large number of MUs that can be used to systematically examine the MU structural change in stroke survivors. From a clinical perspective, the noninvasive nature and efficiency of the methods afford us the ability to develop a diagnostic tool for muscle weakness post-stroke.

REFERENCES


